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## Diagnosis and management of feline diabetes mellitus (Part I)

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Diabetes mellitus is a common endocrinopathy in cats. Approximately 80% of cats may suffer a type 2 like diabetes based on clinical characteristics and islet histology. Type 2 diabetes is a heterogeneous disease due to a combination of impaired insulin action (insulin resistance) and  $\beta$ -cell failure. For both defects to develop environmental as well as genetic factors play a role. The latter have not been characterized in the cat so far, the most convincing evidence on the existence of genetic factors has derived from studies in Australia and the UK in the Burmese cat, in which the frequency of diabetes was shown to be clearly higher than in domestic cats.<sup>1,2</sup> Besides being a Burmese cat other risk factors are: increasing age, male gender, being neutered, physical inactivity, glucocorticoid and progestin administration and obesity. Similar to humans the most important risk factor is obesity and it has been shown that obese cats are 3.9 times more likely to develop diabetes compared to optimal weight cats.<sup>3</sup> Other specific types of diabetes (formerly called secondary diabetes) account for approximately 20% of cases and include pancreatitis, hyperadrenocorticism, hypersomatotropism and the application of progestins or glucocorticoids.

Diabetes typically occurs in middle-aged to old cats, with a strong sex predilection for males. Approximately 60% of diabetic cats presented to our hospital are mildly to severely overweight. Most diabetic cats have the classical symptoms of diabetes, namely polyuria, polydipsia, polyphagia and weight loss. About 10% have overt symptoms of diabetic neuropathy, manifested as hind limb weakness, decreased ability to jump and plantigrade posture. Cats are prone to stress hyperglycemia which has to be differentiated from hyperglycemia due to diabetes by repeated blood glucose measurements or by measurement of fructosamine. Further work-up should clarify the severity of diabetes and the presence of concurrent disease (e.g. stomatitis, urinary tract infection). Treatment should be initiated immediately and include twice daily insulin application (e.g. Caninsulin®, Lantus®) and high-protein-low-carbohydrate diet. Initial dosage (Caninsulin, Lantus) in cats weighing < 4 kg is 1 U/cat given twice daily, cats weighing > 4 kg usually receive 1.5 – 2.0 U/cat twice daily. In cats which have a blood glucose concentration < 20 mmol/l at the time of diagnosis, no more than 1 U/cat twice daily independent of the body weight is given.<sup>4,5</sup>

The cat is a true carnivore which distinguishes it clearly from the omnivorous dog. Using low-carbohydrate-high-protein diet may result in better clinical control and may contribute to diabetic remission. Time of feeding relative to the insulin administration does not seem to play a role, the feeding schedule should, however, be consistent from day to day, either two equal size meals around the time of insulin administration or “nibbling” throughout day and night. Frequent re-evaluations and amendments of insulin dosage are essential to achieve a good glycemic control and to increase the chance of diabetic remission. In our hospital re-evaluations are performed 1, 3, 8, 12 weeks after diagnosis and then approximately every 4 months. Ideally, fasting blood glucose (before the insulin injection) should be between 12-15 mmol/l and the nadir 5-9 mmol/l. Dosage changes are performed with increments of 0.5 U/cat BID approximately every 5 – 7 days.

The owner should assess his animal with regard to the clinical signs of diabetes mellitus on a daily basis. Body weight should be taken at least once a week. It is important that the owner is familiar with the clinical signs of the most important complications of diabetes mellitus, i.e. hypoglycemia and diabetic ketoacidosis. In general, single measurements are considered insufficient to assess metabolic control. Blood glucose curves (BGCs) are necessary to evaluate insulin efficacy, glucose nadir, duration of insulin effect, degree of fluctuation and the Somogyi effect. We prefer that owners give insulin and food at home, and then bring the animal to the hospital (within 2 hours) for a BGC.

However, since cats are prone to stress hyperglycemia blood glucose concentrations measured in the hospital may not reflect the true glycemic situation. We therefore recommend to the owners of diabetic cats to perform so-called home-monitoring (HM) of capillary blood glucose and approximately 70% of the owners are willing and able to do this. Usually, we introduce HM 3 weeks after the initial presentation. Capillary blood is obtained from the pinna or paw and glucose is determined with a portable glucometer (currently with the AlphaTRAK, Abbott Animal Health).<sup>6</sup> Depending on the particular cats we recommend to generate a BGC once a week to once a month. Owners can certainly be taught to interpret a BGC; however, we prefer that decisions regarding changes in the insulin dose be made by the veterinarian and therefore, BGCs be sent to the hospital. The interpretation of BGCs generated at home is similar to those generated in the hospital. The glucose concentration should ideally range from 12 – 15 mmol/l before the insulin injection with a nadir from 5 – 9 mmol/l. A low nadir may occur in insulin overdose, overlap of insulin actions and lack of food intake. If the nadir is > 9 mmol/l, insulin underdose, the counter-regulatory phase of the Somogyi effect and technical problems involving the injection of insulin by the owner must be considered. The duration of effect is defined as the time from insulin injection through the glucose nadir until the blood glucose concentration exceeds 12 – 15 mmol/l. If the duration is less than 8 – 10 hours, the animal usually has signs of diabetes, and if the duration is longer than 14 hours and the insulin is given twice daily, the risk of hypoglycemia increases. One of the major advantages of HM is that it enables frequent generation of BGCs, which may be of particular importance in animals that are difficult to regulate. HM has replaced measurement of urine glucose nearly completely in our hospital. We routinely measure the fructosamine concentration during the re-evaluations. It increases when glycemic control worsens and decreases when glycemic control improves. Metabolic control is usually good when fructosamine levels are between 350 and 450 µmol/l, moderate when values are between 450 and 550 µmol/l and poor when levels are > 550 – 600 µmol/l.

In most cats 2 – 3 months are needed until adequate glycemic control is achieved. It is also mostly during the first 3 months that diabetic remission occurs. With the treatment regimen described above the current remission rate in our hospital is approximately 50%. Interestingly older cats have a greater chance of remission than younger cats.<sup>7</sup> Close monitoring is required because if improvement of glycemic control or diabetic remission happen unnoticed serious hypoglycemia may result.

## References

1. McCann TM, Simpson KE, Shaw DJ, Butt JA, Gunn-Moore DA: Feline diabetes mellitus in the UK: the prevalence within an insured cat population and a questionnaire-based putative risk factor analysis. *J Feline Med Surg* 2007; 9(4):289-299.
2. Lederer R, Rand JS, Jonsson NN, Hughes IP, Morton JM: Frequency of feline diabetes mellitus and breed predisposition in domestic cats in Australia. *Vet J* 2009; 179(2):254-258.
3. Scarlett JM, Donoghue S: Associations between body condition and disease in cats. *J Am Vet Med Assoc* 1998; 212(11):1725-1731.
4. Reusch CE, Robben JH, Kooistra HS: Endocrine pancreas. In: Rijnberk A, Kooistra HS (eds.): *Clinical Endocrinology of Dogs and Cats*, 2<sup>nd</sup> ed., Hannover, Schlütersche Verlagsgesellschaft, 2010; 155-185.
5. Reusch CE: Feline diabetes mellitus. In: Ettinger SJ, Feldman EC (eds.): *Textbook of Veterinary Internal Medicine*, 7<sup>th</sup> ed., St. Louis Missouri, Elsevier, 2010; 1796-1816.
6. Zini E, Moretti S, Tschuor F, Reusch CE: Evaluation of a new portable glucose meter designed for the use in cats. *Schweiz Arch Tierheilkd* 2009; 151(9): 448-451.
7. Zini E, Hafner M, Osto M, Franchini M, Ackermann M, Lutz TA, Reusch CE: Predictors of clinical remission in cats with diabetes mellitus. *J Vet Intern Med* 2010; 24(6): 1314-1321.